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Does the Shortened Environmental Symptoms Questionnaire Accurately Represent Physiological Adaptations Following a 10 Day Heat Acclimation Protocol and the Decay Thereafter?

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Does the Shortened Environmental Symptoms Questionnaire Accurately Represent Physiological
Adaptations Following a 10 Day Heat Acclimation Protocol and the Decay Thereafter?

Rachel Marie VanScoy

B.S., Marist College 2014

A Thesis

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Masters of Science Thesis

Does the Shortened Environmental Symptoms Questionnaire Accurately Represent
Physiological Adaptations Following a 10 Day Heat Acclimation Protocol and the
Decay Thereafter?

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ABSTRACT

Does the Shortened Environmental Symptoms Questionnaire Accurately Represent Physiological Adaptations Following a 10 Day Heat Acclimation Protocol and the Decay Thereafter?

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CONTEXT: Heat acclimation (HA) mitigates exertional heat illness risk. HA occurs during a 10-14 day period, and adaptations will decay with the cessation of exercise in the heat. The confirmation of physiological adaptations and decay is seldom plausible in a clinical setting. The modified environmental symptoms questionnaire (ESQ-14) measures symptom severity during exercise in a hot environment and could help verify HA status resulting in an inexpensive, accessible, and easy to use tool for clinicians. **OBJECTIVE:** To determine if the ESQ-14 detects HA induction and decay. **DESIGN:** Randomized controlled, pair-matched design. **SETTING:** Controlled environmental chamber. **PARTICIPANTS:** Fifteen healthy males (mean \pm sd; age, 23 \pm 3 y; height, 179 \pm 6 cm; weight, 73.47 \pm 7.71 kg; percent body fat, 7.6 \pm 4.8%; $\text{VO}_{2\text{max}}$, 55.1 \pm 5.7 ml \cdot kg $^{-1}\cdot$ min $^{-1}$). **INTERVENTION:** Participants completed 10-11 days of HA involving 90-240 min exercise at 45-80% $\text{VO}_{2\text{max}}$ (40°C, 40% relative humidity). Before and after HA, a heat stress test (HST) was completed. HST involved two 60-min bouts of treadmill exercise (45% $\text{VO}_{2\text{max}}$, 2% grade) with a 10 min break between bouts, under similar environmental conditions as the HA protocol. After HA participants were split into no heat exposure (NHE, n=7) or intermittent heat exposure (IHE, n=8) groups and completed a HST every 5th day for 25 days, in either a thermoneutral (NHE) or hot (IHE) environment; both groups completed a final HST (+25d) in a hot environment. **MAIN OUTCOME MEASURES:** ESQ scores, heart rate (HR), rectal temperature (T_{rec}), OMNI scale, and thermal sensation (TS)

scale were measured before exercise (PRE) and immediately post exercise (IPE) for HST. Mixed model ANOVA evaluated ESQ-14 differences. A Pearson's product-moment correlation followed by a linear regression assessed the ESQ-14 with psychological and physiological data correlations. Alpha level was set at 0.05 for all tests. Data are mean \pm standard deviation, and 95% confidence intervals (CI) included where applicable ($M \pm SD$ [95% CI]). **RESULTS:** HA was confirmed by lower IPE HR (mean difference (before HA-after HA)= 16 ± 1 bpm) and T_{rec} ($0.58 \pm 0.02^{\circ}C$) (both $p \leq 0.05$). IPE ESQ-14 score before HA (22 ± 8 [16, 28] and after HA (11 ± 7 [6, 17]) was significantly greater than PRE ESQ-14 score before HA (5 ± 3 [2, 7]) and after HA (5 ± 3 [2, 7]) (all, $p < 0.001$). Importantly, HA elicited a statistically significant mean decrease in delta ESQ-14 scores (IPE ESQ-14 - PRE ESQ-14) compared to before HA ($(10 \pm 6$ [6, 13]), $t(14) = 5.895$, $p < 0.001$). No significant differences were found between groups (IHE and NHE) and day (after HA and +25d), $p = 0.467$. A relationship between T_{rec} ($p = 0.026$), OMNI ($p = 0.005$), and TS ($p = 0.001$) to delta ESQ-14 scores was found after HA. and between T_{rec} ($p = 0.018$), HR ($p = 0.021$), and OMNI ($p = 0.001$) and delta ESQ-14 scores on +25d. Linear regression revealed a correlation between TS after HA ($r^2 = 0.636$, $p = 0.001$ [95% CI, 4-11]) and OMNI on +25d ($r^2 = 0.589$, $p = 0.001$ [95% CI, 1-4]). **CONCLUSION:** Heat acclimated individuals perceived and incurred less physiological heat stress compared to their pre-acclimated state. The ESQ-14 assessed overall HA induction, through the reduction of symptom severity, and verified some (T_{rec}), but not all (HR) HA induction responses. No difference in ESQ-14 scores when thermal stress was removed or maintained every 5th day. Therefore, the ESQ-14 may not be useful in detecting specific HA adaptations, but is useful in assessing overall HA induction. Further research is needed to assess the ESQ-14 during decay and maintenance of HA.

Key words: Heat acclimation, environmental symptoms questionnaire, decay, maintenance

CHAPTER I: REVIEW OF LITERATURE

Exercise in the heat

Exertional Heat Illness. During prolonged exercise in a hot environment, our bodies may undergo severe thermal strain. Prolonged exercise in the heat can lead to increased cardiovascular strain, decreased performance, and more seriously an increased risk of EHI. Exertional heat illness is a unique problem that laborers, military personnel, and athletes face. Various factors such as equipment/clothing, dehydration, increased body mass index (BMI), and acclimation status negatively impacts the body's ability to thermoregulate. The human body relies on the thermoregulatory system and complex physiological interactions to maintain homeostasis; when this system becomes overwhelmed the potential for EHI increases.

Exertional heat illness encompasses a few different conditions (Table 1); Exercise-associated muscle (heat) cramps, heat syncope, heat exhaustion, exertional heat injury, and exertional heat stroke (EHS).¹ The most severe classification of EHI is EHS, which is characterized as central nervous system (CNS) dysfunction and a body temperature $\geq 40.5^{\circ}\text{C}$ (104.5°F) as a result of prolonged exercise in a hot environment.¹⁻⁴ This occurs when the thermoregulatory system becomes overwhelmed and is no longer able to dissipate heat, resulting in a continual rise in body temperature. If left untreated or improperly treated, EHS can be fatal. The high body temperature can lead to tissue damage and multi organ failure, which is often the cause of death. While EHS occurrence is not entirely preventable, mortality caused by EHS is 100% preventable.

Table 1. Distinction of Exertional Heat Illnesses. (Modified from Casa et al¹)

Condition	Exercise-Associated Muscle (Heat) Cramps	Heat Syncope	Heat Exhaustion	Exertional Heat Stroke	Heat Injury
Description	Acute, painful, involuntary muscle contractions presenting during or after exercise	Collapsing in the heat, resulting in loss of consciousness	Inability to continue exercise due to cardiovascular insufficiency	Severe hyperthermia leading to overwhelming of the thermoregulatory system	Moderate to severe heat illness characterized by organ and tissue injury
Physiologic cause	Dehydration, electrolyte imbalances, and/or neuromuscular fatigue	Standing erect in a hot environment causing postural pooling of blood in the legs	High skin blood flow, heavy sweating, and/or dehydration, causing reduced venous return	High metabolic heat production and/or reduced heat dissipation	

Exertional heat illness poses the greatest risk for athletes during the first 2-3 weeks of preseason.¹ Most occurrences take place in the month of August and are widespread throughout the United States.⁵ Since 1980 the military has seen a decrease in injury and hospitalization from EHI, however, there has been an increase in EHS hospitalization.⁶ The author suggested that the decrease in injury and hospitalization from EHI was due to a heat illness prevention programs that includes HA programs and fluid and electrolyte replacement.⁶ The need to prevent EHI is still relevant as can be seen by the increasing numbers of EHS occurrence.

Prevention. The prevention of EHI can take place through, preseason screening of athletes who may be at greater risk, maintaining hydration status, proper work to rest ratio, modifying activity based on Wet Bulb Globe Temperature (WBGT), knowing the signs and symptoms of EHI, heat acclimation (HA), and having an emergency action plan in place.^{1,2,7} The National Athletic Trainers' Association¹ provides recommendations for the recognition, treatment, and prevention for athletic trainers and health care providers to mitigate the risk of

EH1, such as a 10-14 day heat acclimatization period. Heat acclimatization is a valuable tool that allows an individual to gradually introduce heat exposure that results in physiological adaptations. Heat acclimation is accomplished through the progression of increasing intensity and duration of physical activity. In order to understand HA one must first understand how the thermoregulatory system functions to maintain homeostasis when performing in hot environmental conditions.

Thermoregulation

The thermoregulatory system functions to maintain body temperature through a series of complex interactions involving the CNS, cardiovascular system (CVS), and the integumentary system. These three systems work together to maintain body temperature of $\sim 37^{\circ}\text{C}$, a temperature known as the set point during rest. In the event that body temperature rises various mechanisms will be initiated to dissipate heat in order to return back to 37°C .

Central Nervous System. The thermoregulatory system is controlled through the preoptic area of the hypothalamus (POAH), which is responsible for maintaining equilibrium. The POAH can detect minor changes in central and peripheral temperature. Within the POAH are specific neurons sensitive to preoptic temperature; warm-sensitive, cold-sensitive and temperature-insensitive neurons.⁸ Warm-sensitive neurons account for $\sim 30\%$, whereas cold-sensitive neurons account for $<5\%$ of preoptic neuron population.⁸ Rise in central and peripheral temperature increases the firing rate of warm-sensitive neurons in order to elicit heat dissipation responses, such as sweating.⁸ Furthermore, warm-sensitive neurons inhibit cold-sensitive neurons when preoptic temperature decreases.⁹ Initiation of heat dissipation is vital in thermoregulation allowing for humans to survive in extreme environments.

The central nervous system works along-side the peripheral nervous system through a feedback-loop mechanism, responding to afferent inputs with effector responses. These responses result in the dilation of cutaneous blood vessels, constriction of splanchnic blood vessels, and sweat gland activation, as well as heart and endocrine response; thus impacting the rate of heat storage by allowing the body to dissipate metabolic heat production.

A high body temperature or ‘critical core’ temperature has been suggested to be the limiting factor during performance in a hot environment.¹⁰ González-Alonso et al¹⁰ investigated time to exhaustion in endurance trained athletes with different rates of heat storage, and found that all participants fatigued at an identical body temperature (40.7 – 40.9°C). Participants with the slower rate of heat storage were able to perform longer, however, they fatigued at the same critical temperature as the participants with a higher rate of heat storage. This suggests that time to exhaustion in a hot environment is inversely related to the rate of heat storage.

Researchers have investigated the cause of the development of fatigue as well as, which factors lead to decreased performance during prolonged exercise in a hot environment.¹⁰⁻¹² Elevated body temperature has been shown to be the limiting factor in the ability to perform in a hot environment. One study¹² observed maximal voluntary contraction between men exercising on a cycle ergometer in a hot (40°C) or thermoneutral (18°C) environment until exhaustion. Esophageal temperature reached 40.0 ± 0.1 °C in the hyperthermic group and 38 ± 0.1 °C in the control group. The findings revealed no difference in force generation between groups; however, maximal voluntary force was significantly lower in the hyperthermic ($54 \pm 7\%$) group compared to the control ($82 \pm 6\%$). This proposes that maximal voluntary force was reduced by “central fatigue”, caused by hyperthermia. Demonstrating that fatigue is caused by the central nervous system instead of altered muscle activity as a result of hyperthermia.¹¹

Ratings of perceived exertion (RPE) is a common subjective assessment used in research to assess an individual's effort. One investigator¹¹ found that RPE showed no correlation to EMG measures, however, RPE was found to correlate with esophageal temperature. As body temperature increases, participant RPE increases, thus an individual may feel that they are working harder when there is no increase in exertion.

Cardiovascular System. During moderate-to-heavy exercise in a cool environment a progressive decrease in stroke volume (SV), central blood volume, blood pressure, and right atrial mean pressure, with a concomitant rise heart rate (HR) will occur to maintain cardiac output; a phenomenon known as “cardiovascular drift.”¹³ This phenomenon is more pronounced during moderate-to-heavy exercise in a hot environment, and can be exacerbated by dehydration.¹⁴

González-Alonso et al¹⁴ observed cardiovascular response in hyperthermic endurance athletes while in a euhydrated hyperthermic state (esophageal temperature 39.3°C), hypohydrated state (~4% body weight loss, esophageal temperature 38.1°C), and a state of combined hyperthermia and hypohydration (~4% body weight loss, esophageal temperature 39.3°C) during moderately intense exercise. The results showed that hyperthermia alone decreased SV ($8 \pm 2\%$) and increased HR ($5 \pm 1\%$), whereas hyperthermia with dehydration decreased SV ($20 \pm 1\%$) and increased HR ($9 \pm 1\%$) to a greater degree. The hypohydrated state was identical to the euhydrated hyperthermic state. The combined hypohydrated and hyperthermic group were unable to maintain cardiac output; placing a greater strain on the cardiovascular system, as shown through elevated HR and decreased SV. Thus cardiovascular drift was exacerbated by hyperthermia with the addition of hypohydration during exercise. This will ultimately decrease an individual's ability to thermoregulate, predisposing them to EHI.

Exercise in a hot environment leads to vasoconstriction of the splanchnic and renal beds, redistributing 600-800 ml/min of blood flow to the skin so that metabolic heat can be eliminated.¹³ Additionally, there is an increase in blood flow to working muscles to keep up with oxygen demands. Consequently the cardiovascular system will compete with the skin for blood flow causing strain on the cardiovascular system. An early review by Rowell¹³ postulated that the redistribution of blood flow to the cutaneous vasculature with a rise in body temperature was a leading factor of cardiovascular drift. Throughout upright exercise in a cool environment muscles work as a pump to return circulating blood centrally; enabling stabilization of central blood volume, SV and central venous pressure.¹⁵ However, during upright exercise in a hot environment cutaneous vasodilation allows for the cutaneous veins to refill faster, not allowing the muscle pump to work effectively resulting in a decrease in SV.¹⁵

In contrast, a more recent review¹⁶ proposed that cardiovascular drift is primarily due to an increase in HR, caused by rising body temperature and sympathetic nervous system activity, as opposed to an increase in cutaneous blood flow. The increase in HR decreases the time for the ventricle to fill, hence a decrease in SV. This notion was supported by a plateau in skin temperature while body temperature continues to rise along with an increase in HR and decrease in SV; thus skin temperature did not contribute to further decline in SV.¹⁰ Exercise in the heat strains the cardiovascular system limiting the regulatory ability to maintain homeostasis.

Integumentary System. In order to maintain homeostasis while body temperature rises, the thermoregulatory system initiates responses within the integumentary system to dissipate heat. These responses include the dilation of cutaneous blood vessels and the activation of sweat glands. The exchange of internal heat to the environment occurs through evaporation, convection, conduction, and radiation; the integumentary system is a primary contender in heat

dissipation. Located within the skin are eccrine sweat glands, which are innervated by sympathetic cholinergic fibers. Water and electrolytes enter the sweat gland from the interstitial fluid and exit through a pore on the surface of the skin, allowing for sweat to evaporate in the surrounding environment. Dilation of cutaneous blood vessels allows for increase in warm blood flow to the skin aiding in the exchange of energy from the warm blood to the external environment (convection, radiation and conduction).

The evaporative mechanism for heat loss (sweating) is strongly influenced by SR and the surrounding environment. Sweat gland secretion is specific to each body region, and begins at a certain body temperature, similar to the thermoregulatory set point. The point of zero central drive for chest sweating occurs at an esophageal temperature of 37.37° C in un-acclimatized individuals.¹⁷ The rate at which sweating occurs during exercise varies among fitness level; unfit individuals will have a lower sweat rate than fit individuals.¹⁷ Thus unfit individuals will experience greater thermal strain due to less efficient heat loss, and be at greater risk for EHI.

Dehydration negatively affects the body's ability to dissipate heat. As previously mentioned, a competition exists for blood between the cardiovascular system and the periphery during prolonged exercise in the heat. This competition is exacerbated by dehydration and ultimately the cardiovascular system will prevail, consequently inhibiting heat dissipation. This was observed in a previous study¹⁸ assessing the influence of hydration on the circulatory and thermoregulatory system in fit individuals during exercise in 35°C while hyperhydrated, hypohydrated or euhydrated. The results showed the threshold for cutaneous vasodilation occurred at a lower skin temperature when hyperhydrated and euhydrated (36.85°C, 36.90°C, respectively), whereas in a hypohydrated state the onset (37.32°C) of heat dissipation was delayed. In a hypohydrated state esophageal temperature continued to rise while forearm blood

flow was ~50% less than hyperhydrated or control.¹⁸ The author suggested that the cutaneous blood flow decreases in a hypohydrated state to maintain cardiovascular function.¹⁸ Therefore, a decrease in blood flow to the periphery, due to dehydration, will lead to a continuous rise in body temperature, due to the inability to dissipate heat.

Uncompensable Heat Stress. When body temperature increases, the mechanisms described above are initiated to dissipate heat. However, the thermoregulatory system can become overwhelmed where heat storage becomes greater than heat dissipation. Consequently, body temperature will continue to rise to dangerous levels; known as uncompensable heat stress. Various factors can inhibit heat dissipation: larger BMI, poor CV physical fitness, equipment and clothing, environmental conditions, lack of HA, pre-existing illness, fatigue, sleep loss, and previous history of EHS. A HA protocol can allow the body to physiologically adapt and overcome uncompensable heat stress in many scenarios, in order to protect the thermoregulatory system from becoming overwhelmed.

Heat Acclimation

The human body has the ability to physiologically adapt to various climates, such as cold and hot temperatures, and to various altitudes. Repeated exposure to such conditions will produce changes that reduce the physiological strain placed upon various systems in the body a phenomenon termed acclimation; this allows humans to survive and perform in extreme environments. For instance, HA is a process where the body adapts in order to maintain homeostasis in a warm environment by decreasing cardiovascular and thermal strain, and improving exercise tolerance. For this reason HA is a vital component in the prevention of EHI, and is widely considered the most powerful acute human adaptation. It is important to understand the difference between heat acclimation and heat acclimatization; *Heat acclimation*

occurs in an artificial environment (i.e., environmental chamber), whereas, *heat acclimatization* occurs in a natural environment (i.e., outdoor exercise in the heat).

Time Course of Induction. The induction of HA occurs over a 10-14 day period involving repeated stress that disrupts homeostasis.^{19,20} The physiological adaptations that occur are decreased HR, RPE, rectal temperature (T_{rec}), sweat and renal Na^+ and Cl^+ concentrations, coupled with plasma volume (PV) expansion and an increase in SR.²⁰ Within 4-6 days two-thirds to 75% of adaptations occur.²¹ Furthermore it takes 10-14 days for adaptations to reach 95% of their maximal adjustment, with each adaptation occurring at various points throughout a HA period.²⁰ Garret et al,²² found that during short term (5 days) HA adaptations included improved exercise capacity, decreased rectal temperature and decreased HR, however an increase in PV was not shown. In order to gain complete physiological adaptations of HA it is recommended that an individual follow a 14 day protocol.^{1,20}

Two methods that have been used in laboratory settings to induce heat acclimation are traditional exercise heat exposure¹⁹ and controlled hyperthermia.²³ Traditional exercise heat exposure consists of a controlled workout procedure. The stimulus for the physiological adaptations during this method encompasses wet and dry bulb temperature, radiation flux, clothing and anthropometric measures.²³ However, Fox et al²³ found that traditional method had limitations in this design that would inhibit maximal adaptations. Such as the nature of the protocol, as individuals would perform at the same intensity, ultimately reaching habituation.²³ Since the thermal strain remains constant the body's ability to maintain homeostasis improves and eventually the thermal stimulus will decrease if intensity is not modified.²³ This idea led to the development of stimulating acclimation through a controlled hyperthermia design, accomplished by controlling body temperature at 38.5°C. By controlling the body temperature,

with an ever changing exercise stimuli, you are able to prevent accommodation to the exercise and thus allow a constant thermal stimulus for adaptation.¹⁹

Adaptations. The first adaptations to occur are of cardiovascular origin (decreased HR and RPE and an increase in PV). Plasma volume begins early however the expansion is transient and will begin to decay around day 8-10.^{19,20} Plasma volume has shown to increase by ~6% after heat acclimation resulting in an increase in cardiac output of ~4.5%.²⁴ Increased plasma volume accommodates the redistribution of blood flow from the splanchnic bed to working muscles and cutaneous vessels, as previously discussed. This will allow the body to maintain cardiac output during heat exposure and reduce the cardiovascular strain. Hence a decrease in syncopal episodes during the first few days of HA to allow further adaptation to occur.²⁵ Once other adaptations occur, however, expanded plasma volume is of less importance, and naturally declines back to pre-acclimation levels.

During the initial days of HA heart rate will begin to decrease stabilizing cardiac output in conjunction with an increase PV. Pandolf et al²⁶ observed physiological responses in 24 male participants during a 9-day HA protocol and found that HR decreased significantly from a mean value of 160 beats per minute for day 1 to 124 beats per minute for day 9. More importantly, this study did not find significant changes for HR on days 7-9. Therefore the full scope of cardiovascular adaptation occurs earlier in the HA process.

Around days 5-8 of HA rectal temperature decreases to further reduce thermal strain. A reduction in body temperature is an important variable as it shows that the demands for heat loss are being met with equivalent heat dissipation. Body temperature rises with exercise but will ultimately plateau as a balance forms between metabolic production and heat loss. Physical fitness also plays a role in the rate of adaptation. One study found that, during a 9d traditional

method of HA, the least fit participants ($\text{VO}_{2\text{max}}=35.5$ and $40.5 \text{ ml kg}^{-1} \text{ min}^{-1}$) took 8 days for T_{rec} to plateau, whereas the most fit participants ($\text{VO}_{2\text{max}}= 63.8$ and $58.6 \text{ ml kg}^{-1} \text{ min}^{-1}$) took 4 days to reach plateau.²⁶ Therefore the process of HA is not uniform between all individuals.

Last to occur is a change in the sweat response to heat exposure. Not only does the sweat rate increase during HA but the point of zero central drive is reduced. The ability for the body to eliminate internal metabolic heat improves during the course of HA, which is evident in the improvement of sweating; an adaptation that occurs in the latter portions of a HA protocol. Nadel et al¹⁷ observed thermal responses during a 10 day heat acclimation protocol in dry (45°C) and humid (36°C) conditions at $50\% \text{ VO}_{2\text{max}}$. The findings showed a 0.24°C reduction in the point of zero central drive after heat acclimation compared to pre HA. In addition, the SR was higher after acclimation compared to pre HA. In a HA state sweating will occur sooner and increase with rise in body temperature, allowing for internal body heat to dissipate sooner than un-acclimated individuals, reducing the thermal load. As shown above HA improves the body's ability to dissipate heat and decreases the thermal strain.

Time Course of Decay. Adaptations are only transient and in the event that thermal stimulus is removed, decay of adaptations will begin. Decay of each adaptations occurs at various rates based on days of heat exposure, length of HA, natural HA, type of training during HA, and individual characteristics (i.e. fitness level).^{19,20} Decay can be calculated using a formula modified by Pandolf et al²⁶. This formula determines the percentage lost of a physiological adaptation (i.e., T_{rec} , HR, SR).

$$\%Loss = \frac{\text{Average value after day of decay} - \text{Average acclimation value}}{\text{Average unacclimated value} - \text{Average acclimation value}} \times 100$$

The first adaptation to decay is of cardiovascular origin. Heart rate decays at a faster rate than rectal temperature, as shown in table 2. Within three weeks of removing thermal stimulus

heart rate and sweat rate return to pre-acclimation values, at the same time rectal temperature will be half of the pre-acclimation value.²⁷ Pandolf et al²⁶ assessed physiological variables (T_{rec} and HR) 3, 6, 12, and 18 days after HA, illustrated in table 2. The small percentage of loss over 18 days was attributed to the fact that participants had an above average level of fitness (49.5 ml $\text{kg}^{-1} \text{min}^{-1}$). As previously mentioned physical fitness plays a role in the rate of adaptation; This is also true for the decay of HA, however the rate will be slower for physically fit compared to less fit individuals.

Table 2. The percentage loss (positive number) or gain (negative number) of adaptations following heat acclimation.

Study	Variable	Day post heat acclimation			
		6-9	12-16	18-23	26-28
Pandolf et al ²⁶	Rectal temperature	13	18	4	-
	Heart rate	23	20	29	-
Weller et al ²⁸	Rectal temperature	-	15	-	-9
	Heart rate	-	33	-	27
Williams et al ²⁷	Rectal temperature	25	40	50	-
	Heart rate	50	80	100	-

Weller et al²⁸ found that re-acclimation was accomplished after 2 days and 4 days of heat exposure following 12 and 26 day without heat exposure, respectively. This suggested that once HA was attained individuals could spend as long as a month in a cooler environment and that reduction of acclimation would not be as extensive. In comparison Williams et al²⁷ recommended that one week without heat exposure would require one day of re-acclimation before returning to physical activity in a hot environment. The ability to maintain physiological adaptations is extremely important for physically active individuals that plan to return to a hot environment after a break.

Practical Application. Heat acclimatization is most importantly a prevention method for EHI. By creating physiological adaptations, the body will become better at maintaining homeostasis. The reduced risk of EHI is a result of decrease in thermal strain (i.e., decrease HR,

body temperature, increase sweat). Not only is HA a preventative measure it also improves performance and fitness levels. Heat acclimation has shown to increase VO_{2max} by 8% and improve time trials by 8%.²⁴ Therefore, implementation to improve physical fitness HA can be implemented in a training program.

The importance of understanding HA decay is to determine how to maintain physiological adaptations when heat exposure is unavailable. This is appropriate for athletes traveling for competition or military deployment. Monitoring an individual through HA, however, isn't always as accessible. Rectal thermometers are the gold standard for measuring body temperature, but these measures can be invasive and costly.¹ Monitoring HR is easily assessable and minimally invasive but it is only one portion of the puzzle. Researchers have developed a subjective questionnaire to measure symptoms related to EHI; coaches, parents, athletes, laborers, and military personnel could easily use this tool to monitor the severity and frequency of symptoms.

Environmental symptoms questionnaire

Development. The environmental symptoms questionnaire (ESQ) was developed by Kobrick and Sampson²⁹ to assess the severity of symptoms during various extreme environmental conditions, specifically acute mountain sickness. Several versions of the ESQ have been developed to improve assessment of symptoms, shown in table 3. At first, the ESQ was comprised of 52 questions rated on a scale of 1-9 (1 slight, 5 moderate, 9 severe) but was later changed to ultimately include 68 questions rated on a scale of 0-5 (0 not at all, 1 slight, 2 somewhat, 3 moderate, 4 quite a bit, 5 extreme).²⁹⁻³² The questionnaire included a large range of symptoms to insure no symptoms were excluded, as symptoms vary among individuals.

Table 3. Evolution of the Environmental Symptoms Questionnaire (ESQ).**Abbrev: EHI, exertional heat ailment; HA, heat acclimation; SHI, Subjective heat illness.**

ESQ-I ²⁹	52 questions. Yes/No responses followed by a scale of 1-9 (1- slight, 5-moderate, 9-severe).
ESQ-II ³⁰	56 questions. Removed yes/no format. Scale of 0-5 (0- not at all, 1-slight, 2- somewhat, 3-moderate, 4- quite a bit, 5- extreme). Procedures for administration and scoring.
ESQ-III ³¹	67 questions (added based on symptoms reported within acute mountain sickness research). Detailed recommendation for administration and scoring.
ESQ-IV ³²	68 questions (Adding “I’m hungry”) Rewording items for the past tense.
SHI ³³	22 symptoms extracted from the ESQ based on relevance to clinical EHI.
ESQ -14 (a.k.a. Modified ESQ) ³⁴	Included signs and symptoms of EHI based on previous research. ³⁵
ESQ-12 ³⁶	Created using highest reported mean scores and responses of the ESQ-14 and ESQ-II throughout HA.

Researchers revealed five symptom clusters using the ESQ-II and developed a scoring system in order to reduce response error and bias; Exertion (49.7%); Fatigue (17.6%); Ear, eye, nose, and throat (13.3%); Headache and Nausea (10.3%); and Wellness (9.1%).³⁰ Further investigation of the ESQ-III resulted in expanding the symptom clusters, from five categories to nine, in order to describe the patterns within the data during analysis; Cerebral AMS (12.8%); Respiratory AMS (17.1%); Ear, nose, throat (9.1%); Cold Stress (12.9%); Distress (11.0%); Alertness (8.4%); Exertion Stress (8.6%); Muscular discomfort (8.8%); Fatigue (11.3%).³¹

Researchers can use the ESQ in extreme environmental conditions (heat or cold) by selecting a subset of questions that relate to specific condition (hypo or hyperthermia) without weighting individual items. Johnson and Merullo³³ extracted 22 symptoms from the ESQ-IV to develop an index of subjective heat illness (SHI) during a heat acclimation protocol. This is the first study, to our knowledge, that has modified the ESQ to track the symptoms of heat stress during heat acclimation. Further modification has occurred in order to shorten the ESQ to be more specific to

heat stress; these modifications are based on clinical symptoms of EHI³⁵ as well as research findings.^{34,36}

Validity. The early ESQ's have shown to be a valid and reliable technique in the assessment of acute mountain sickness.²⁹⁻³¹ Further exploration using shortened ESQ's for AMS and heat stress have also shown to be a valid tool in research.^{34,36,37}

Yamamoto et al³⁴ analyzed the correlation between the ESQ-14 and the ESQ-II during four 12 km trail runs over a 14 day period. The findings showed that the ESQ-14 was highly associated with the ESQ-II before ($r=0.80$) and after ($r=0.86$) trail runs. This suggests that the ESQ-14 is a valid source in determining the symptom severity during exercise heat exposure. Additional research found that the ESQ-14 and ESQ-12 correlates with the ESQ-II during a 10 day heat acclimation period.³⁶ Stearns et al³⁶ further assessed the correlation of the ESQ (ESQ 12, 14 & II) to primary physiological markers of HA (HR, T_{rec} , RPE). No correlation was found between the three ESQ's and RPE. However, the ESQ-12 showed to be correlated with HR ($r=.430$, $r^2=0.185$) and T_{rec} ($r=0.400$, $r^2=0.160$) whereas the ESQ-II and ESQ-14 were only correlated with HR ($r=0.424$, $r^2=0.180$; $r=0.353$, $r^2=0.124$, respectively). No previous research has assessed the correlation between the ESQ and SR.

Previous Research. Numerous studies have assessed subjective responses during exercise and physical activity in hot environmental conditions using the ESQ; allowing researchers to quantify psychological responses during thermal strain.

Uniform/equipment and clothing can inhibit heat dissipation that will result in increased risk of EHI. As the risk of EHI increases it is natural to think that subjective responses and symptoms of EHI will also increase. This was shown in one particular study where researchers found that the severity and frequency of symptoms reported on the ESQ-IV was increased when

military personnel wore impermeable chemical protective clothing versus battle dress uniform in hot conditions (35°C (95°F) and 60% RH).³⁸ This notion was further supported by a study assessing the perceptual responses while wearing a football uniform in the heat (33°C (91.46°F) and 48% to 49% RH).³⁹ They found that wearing full or partial uniform increased after exercise ESQ scores compared to no uniform. Decreasing the ability to dissipate heat will increase thermal strain, as previously discussed, hence an increase in perceptual responses.

Researchers have also investigated the most commonly reported symptoms related to heat illness during trials in hot conditions.^{33,36} The symptoms most commonly reported during trials inducing thermal strain are feeling sweaty, warm and feeling weak.^{33,36,38} Increases in symptoms or the severity of symptoms are a sign that an individual may be at greater risk for EHI. Table 4 represents the average individual symptom severity reported during exercise in the heat. The frequency of signs and symptoms related to EHI have shown to decrease during the course of heat acclimation in sequence with reduced physiological strain.³⁵

Table 4. Top reported symptoms and average scores.
Abbrev: ESQ, Environmental symptoms questionnaire.

Johnson et al ³⁹		Kobrick et al ³⁸		Stearns et al ³⁶			
ESQ		ESQ-IV		ESQ-56		ESQ-14	ESQ-12
"I feel warm"	3.5	Sweating all	4.4	Sweating	3	"I feel thirsty"	3
"I feel	2.5	over		Warm	3	"I feel hot"	3
lightheaded"		I felt warm	4.0	Happy	3	"I will play at	2
"I feel weak"	2.4	I had a	3.4	Thinking clear	3	my best"	Weak
"It is hard to	2.4	headache		Well	3	"I feel tired"	2
breathe"		I was short of	2.6			"I feel	1
"I feel faint"	2.3	breath				lightheaded"	1
		It was hard to	2.5				
		breathe					

Various studies have used the ESQ to assess symptom severity during HA as shown in table 5.^{33,34,36,40,41} Previous investigation examining the symptoms of heat illness found that the number of symptoms being reported was significantly higher during the first 2 days of HA than

the remaining 8 days.³³ Similar findings were shown using the ESQ-14, where post exercise scores were elevated on days 1 and 2 compared to subsequent days of HA.⁴¹ Stearns et al³⁶ found a significant effect of time for ESQ-II, ESQ-14, and ESQ-12. However, there was no significant difference between days and this could be due to limitations within the study (i.e., small sample size). Upon further analysis they found large effect sizes of post-exercise ESQ scores for day 1 vs. day 4, 7, and 10. This indicates a clinically relevant change between ESQ scores during the first few days of HA to the latter portion of HA. The first few days of HA provide the greatest strain on the cardiovascular system before physiological adaptations (decrease in HR, RPE, and an increase in PV). The ESQ findings during HA appear to fall in line with adaptations that reduce cardiovascular strain, which occur between days 3 and 6.

It is important to note that no previous study, to our knowledge, has been conducted using the ESQ during the decay and maintenance of heat acclimation. It begs to question whether symptoms increase as the adaptation of physiological variable decays back to pre acclimation levels.

Table 5. The evolution of the environmental symptoms questionnaire (ESQ)

Abbrev: HA, heat acclimation; SHI, Subjective heat illness; RH, relative humidity; WBGT, Wet bulb globe thermometer.

Study	ESQ	Method	Environment	Results
Johnson et al ³³	SHI	4-g vs. 8-g dietary salt groups. 10 d HA protocol. ESQ measured post heat exposure. 8 hours of heat exposure/d	41°C, 20% RH	4-g salt group reported more heat illness on the first 2 days compared to 8-g salt group. SHI was higher during the first 2 days of HA compared to subsequent days.
Yeargin et al ⁴⁰	Modified ESQ	8 d preseason practice session; d 1-5, and 7 were 1 practice/d; Day 6 and 8 had 2 practices each d.	21.8-32.1°C dry bulb, 57.7-92.0% RH, and 21.9-30.1°C WBGT	Post practice ESQ total score was significantly higher than the pre practice total scores. No difference between days was found.
Yamamoto et al ³⁴	ESQ-II and ESQ-14	Four, 12 km trail runs within a 14 d period.	WBGT 26.5°C	Strong correlation between ESQ-II and ESQ-14 before and after trail run.
Yeargin et al ⁴¹	ESQ-14	10 d preseason practice sessions; football drills, contact hitting, conditioning, and education (2.8 ± 0.5 hours)	Average maximum WBGT 23±4°C	ESQ scores significantly increase post practice. No pattern for ESQ across all days. ESQ scores were elevated on days 1 and 2.
Stearns et al ³⁶	ESQ-II, ESQ-14, ESQ-12	10 d HA protocol. 3.5 mph, 5% grade for 90 minutes. Pre and post ESQ measures.	33 °C, 30-50% RH	ESQ-II, ESQ-14 AND ESQ-12 Post scores were greater on day 1 vs. 4, 7 and 10 (significant effect of time).

Conclusion

Heat acclimation is a valuable method of preventing EHI. Implementing a HA protocol can be done within various settings (i.e., school athletics and military), however it is important to know when full adaptations have been reached and when these adaptations begin to decay with the absence of heat exposure. This can be accomplished by monitoring HR, rectal temperature, SR and perceptual scales during HA (i.e., ESQ, RPE). Observing primary physiological markers

(T_{rec} , HR, SR) allow coaches, athletic trainers, athletes, military personnel, etc. to track HA; however, these techniques can become invasive, costly and difficult to use.

To our knowledge no previous research has investigated the effectiveness of the ESQ to track the decay of HA. It is vital to understand how the ESQ can assess symptoms of heat exposure, specifically during the induction and decay of HA. It is important to determine when adaptations are being gained and lost to allow an individual to know when the risk of EHI may increase. Signs and symptoms that are reported may be a warning sign of EHI.

Further research is needed in order to assess how the ESQ can be used as a tool as a prevention measure for EHI. Reporting of symptoms allows for intervention prior to a medical emergency. Athletes may not report symptoms to medical personnel, thus by using the questionnaire it will provide an opportunity to take action if symptoms are increasing (i.e., altering rest to work ratio, providing fluids, cooling, etc.).

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CHAPTER II: INTRODUCTION

Performing physical activity in extreme environmental conditions (i.e., hot, cold, and various altitudes) produces physiological stress within the human body. Prolonged exercise in a hot environment increases risk of exertional heat illness (EHI), however heat acclimatization (HA) has been used to mitigate the possibility of EHI. Heat acclimatization is a process where the body physiologically adapts in order to maintain homeostasis by decreasing cardiovascular and thermal strain, and improves exercise tolerance. Physiological adaptations occur gradually over a 10-14 day period and include changes such as, decreased heart rate (HR), body temperature and an increase in sweat rate (SR).^{1,2} Through these adaptations the body will be able to maintain homeostasis and decrease thermal strain. Not only is HA a preventative measure, it also improves performance and fitness levels.³ In the case that heat exposure is removed the decay of these adaptations will occur. Currently clinicians do not measure physiological variables to verify an individual has fully adapted following a heat acclimatization protocol.

The environmental symptoms questionnaire (ESQ) was developed by Kobrick and Sampson in 1979⁴ to assess the severity of symptoms during various extreme environmental conditions, specifically acute mountain sickness. However, through modification and refinement the ESQ can be used within hot conditions to track the perceptual response of thermal strain. The ESQ is a series of likert scale-based questions originally rated from 1-9 (1 slight, 5 moderate, 9 severe) but later changed to 0-5 (0 not at all, 1 slight, 2 somewhat, 3 moderate, 4 quite a bit, 5 extreme).⁴⁻⁷ This allowed for ease of use by participants and researchers. More recent modification resulted in the development of the ESQ-14, which is used specifically to assess hyperthermia.⁸ The ESQ-14 has been used within heat research^{8,9} and previous investigation

found the ESQ-14 to correlate with HR, however it did not show correlation with rectal temperature (T_{rec}) or perceptual measures.⁹ Currently, to our knowledge, no research exists exploring the relationship of the ESQ-14 to skin temperature (T_{sk}) during exercise in a hot environment.

The most reported symptoms on the ESQ during thermal strain are feeling sweaty, warm, and weak.⁹⁻¹¹ As the process of HA takes place the physiological adaptations reduce the thermal and cardiovascular strain. Previous research has shown a reduction of symptom frequency and severity after the first few days of HA compared to subsequent days.^{9,12,13} The ESQ, however, has not been analyzed during the maintenance and decay of HA.

The ESQ-14, if shown to be sensitive in detecting changes during and after HA, can be of assistance to the military, laborers, and athletes. This indirect measure could help verify HA, to ensure the risk of EHI is decreased, and to determine when the risk develops again with the cessation of activity in a hot environment. Compared to other costly and invasive devices (i.e., rectal thermometer and ingestible thermistor) the ESQ-14 is cheap, accessible, and easy to use.

Therefore, the purpose of this study is to determine if the ESQ-14 is sensitive enough to detect change in HA status during induction and decay of HA and to determine the correlation with physiological and perceptual markers. This is the first study to evaluate the ESQ-14 during the decay and maintenance of HA. We hypothesize that the ESQ-14 total scores will be decreased after HA compared to before HA. We also hypothesize that the ESQ-14 scores will increase if HA status is lost follow the cessation of exercise in a hot environment and that ESQ-14 scores will be comparable to post HA following a heat exposure every 5th day.

CHAPTER III: METHODS

Participants

Eighteen healthy recreationally active male participants volunteered for this study. Inclusion criteria included: chronic health problems, exertional heat stroke within the past 3 years, and no history of cardiovascular, metabolic, respiratory disease or musculoskeletal injury that limits physical activity. Participants were excluded if they exercised less than once per week or had below average aerobic fitness ($\text{VO}_{2\text{max}} < 45 \text{ ml}\cdot\text{kg}\cdot\text{min}^{-1}$). Female participants were excluded from this study due to the influence of the menstrual cycle on resting body temperature.¹⁴ The study took place between October and March in the northeastern part of the United States to decrease the chance of participants being heat acclimatized; average conditions $10.4 \pm 2.2^\circ\text{C}$ and $70.5 \pm 6.5 \%$ relative humidity (RH). The university's institutional review board approved the methodology and written informed consent was obtained prior to data collection.

Experimental design and overview

This study was part of a larger study assessing the effectiveness of a periodic exercise-heat exposure intervention after HA; methods, data, and results published elsewhere. Data from the randomized control study were used to assess the ESQ-14 during heat exposure before and after HA. The study design is illustrated in figure 1. During baseline testing participants were pair matched by $\text{VO}_{2\text{max}}$, exercise history and body surface area. Participants complete a 10-day HA protocol and once heat acclimated were then divided into two groups, no heat exposure (NHE) and intermittent heat exposure (IHE). All participants completed the same HA and heat stress test (HST) protocol. Heat stress test were performed before and after the HA protocol in order to confirm HA was effective and provide evidence of adaptation to heat stress. After HA

participants completed four HST in either a hot (IHE; n=9) or thermoneutral (NHE; n=7) environment every fifth day (+5, +10, +15, +20d). Both groups completed a final HST (+25d) in a hot environment. For the present study, measures from the before HA, after HA, and +25d were analyzed. Two participants were excluded from analysis due to orthopedic injury sustained outside of the study. One additional participant was excluded due to non-compliance with collection of perceptual information. A trial was stopped if one of the following occurred; participants requested to stop, heart rate was > 90% of age predicted max, rectal temperature > 40 °C, uneven or altered gate, signs or symptoms of EHS, or if the maximum exercise time was completed. If a trail was stopped early immediate post measures were taken.

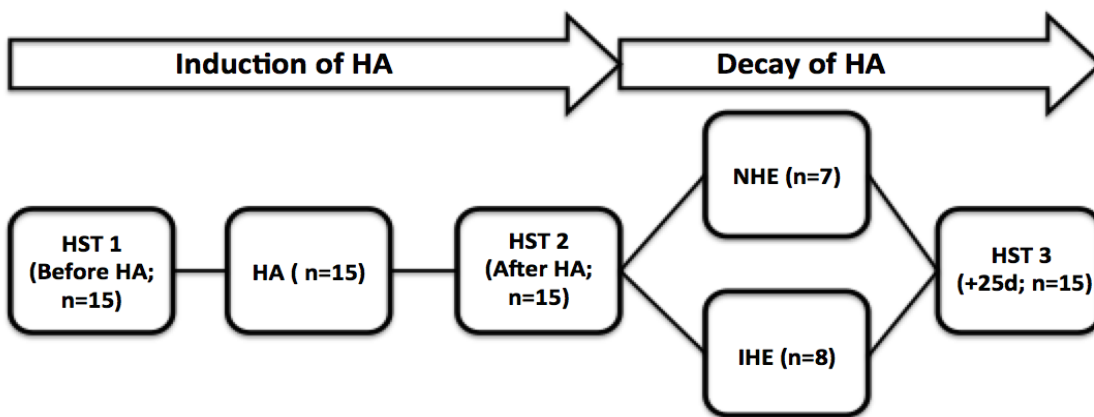


Figure 1. Study design. Abbrev: HST, heat stress test; HA, heat acclimation; NHE, no heat exposure; IHE, intermittent heat exposure.

Baseline testing

Baseline measures included height, body weight and body fat using a wall mounted stadiometer, scale (T51P, Ohaus, Pine Brook, NJ), and skin fold calipers (Lange Skinfold Caliper, BetaTechnology Incorporated, Cambridge, Maryland), respectively. We calculated percent body fat by measuring skinfold at three sites (chest, abdomen, thigh).¹⁵ Next, participants completed a $\text{VO}_{2\text{max}}$ test on a treadmill at 2% grade in a thermoneutral environment. Speed

increased every 2 minutes until the participant reached voluntary exhaustion. Expired gases were collected and analyzed using a metabolic cart (TrueOne® 2400 Metabolic Measurement System, Parvo Medics, Sandy, Utah). Heart rate (Model T5K564, Timex Group USA, Inc., Middlebury, CT) was monitored during the ramping protocol. To confirm participant reached $\text{VO}_{2\text{max}}$ three of the four following criteria had to be met: HR within 10 beat per minute of age predicted max, respiratory exchange ratio ≥ 1.10 , OMNI scale of perceived exertion (appendix A) ≥ 9 , and/or a VO_2 plateau with increased workload.¹⁶

Heat acclimation protocol

Researchers instructed participants to avoid alcohol and strenuous exercise for 24 hours, and caffeine for 8 hours before all testing to reduce any effect on the dependent variables. Heat acclimation consisted of 10-11 days of exercise (90-240 min) in a hot environmental chamber (40°C and 40% RH). Six consecutive days of HA consisted of a 90-minute protocol using a controlled hyperthermia technique to ensure a constant thermal stimulus.^{17,18} Exercise intensity was adjusted so that within 30 minutes T_{rec} reached 38.5°C and remained $\geq 38.5^\circ\text{C}$ for the remaining 60 minutes of the protocol. The remaining days of HA included intermittent exercise on a treadmill at 45-80% of participants $\text{VO}_{2\text{max}}$ for 2 or 4 hours with 10 or 60 minutes of rest breaks, respectively. The variation within the HA protocol is due to additional research purposes published elsewhere.

Heat Stress Test

For each HST, participants performed two 60 minute bouts of treadmill exercise at 45% $\text{VO}_{2\text{max}}$ with a 2% grade. Ten minutes of rest was provided between each bout. Before starting the exercise participants were seated for 20 minutes of rest to allow for physiological variables to stabilize.

Measurements

Upon arrival to the lab participants provided a urine sample to determine hydration status. Urine was analyzed using a refractometer to determine urine specific gravity (USG) (A300CL, Atago, Bellevue, WA). A hydrated state was defined as USG <1.020. If a participant was found to have a USG ≥ 1.020 then they were instructed to drink 500 mL of water prior to the start of exercise. To measure regional T_{sk} , a thermocrom (DS1921G, Embedded Data Systems, Lawrenceburg, KY) was placed on the participant's right chest, deltoid, thigh, and calf using surgical tape. Data collected from the thermocrom was later used to calculate whole body mean T_{sk} .¹⁹ Rectal temperature was measured using a rectal thermometer inserted 10-12 cm beyond the anal sphincter (model 401, Measurement Specialties, Beavercreek, OH). Heart rate was taken using a HR monitor strapped to the chest (RaceTrainer™, Timex, Middlebury, CT). Rectal temperature and HR were recorded before (PRE), every 10 minutes during exercise, and immediately post exercise (IPE).

Pre and post exercise nude body mass was recorded. Water was provided *ad libitum* and consumption was measured and recorded. Body mass loss (BML) was calculated by subtracting post to pre body mass and accounted for urine/fecal losses and fluid consumption. Sweat rate was calculated by dividing BML with exercise time. To determine the percent gain or loss of physiological adaptations the following equation was used.²⁰

$$\%Loss = \frac{\text{Average value} + 25d \text{ of decay} - \text{Average acclimation value}}{\text{Average unacclimated value} - \text{Average acclimation value}} \times 100$$

A positive value indicates loss/decay of adaptation, whereas a negative value indicates increase in HA.

Perceptual measures were taken PRE, every 10 minutes during exercise, and IPE. We used the OMNI scale of perceived exertion to measure participant's effort, which was rated on an

11-point scale and ranged from 0 (extremely easy) to 10 (extremely hard) in 1-point increments.²¹ Additionally, the thermal sensation scale was used, which was rated on a 17-point scale ranging 0 (unbearably cold) to 8 (unbearably hot) in 0.5-point increments (appendix B).²²

A validated ESQ-14 (appendix C)^{8,9} was used in this study to determine symptoms of heat illness. The ESQ-14 contains 14 symptoms; “I feel lightheaded,” “I have a headache,” “I feel dizzy,” “I feel thirsty,” “I feel weak,” “I feel grumpy,” “It is hard to breathe,” “I will play at my best,” “I have a muscle cramp,” “I feel tired,” “I feel sick to my stomach (nauseous),” “I feel hot,” “I have trouble concentrating,” “I have ‘goose bumps’ or chills.” This is a 6-point scale (0-5), ranging from “not at all” (0) to “extreme” (5). The scale was summed based on the rating of “not at all” to “extreme” for symptoms (with the exception of “I will pay at my best,” which is graded in reverse). Participants were instructed on how to use this scale. The ESQ-14 was filled out during all HST protocols after 10 minutes of seated rest prior to the start of exercise (PRE) and IPE. Both measures were taken in the environmental chamber, with the exception of HST performed on +5, +10, +15, +20 after HA for NHE where it was taken in a thermoneutral environment.

Statistical analysis

Outliers were detected using a Q-Q plot and boxplot. An outlier was found for starting HR at one time point. The average starting HR for this participant during all time points was higher than others in his group; therefore, the outlier was replaced with the participants average starting HR over all days. Missing data points for ESQ-14 score IPE (4 data points) and delta (4 data points) score before HA were replaced with group average total at that time point; no other missing data points were replaced.

Data for the analysis comparing before and after HA was pooled. Data comparing HA and +25d was analyzed by splitting participants into groups (IHE and NHE). For the purpose of this study we analyzed before HA, after HA, and 25+d (figure 1). A paired-samples t-test (SPSS, Version 20, Chicago, IL, USA) was used to determine whether there was a statistically significant mean difference between IPE HR, T_{rec} , SR, and exercise time from before HA to after HA. Physiological response data resulting from this study is reported elsewhere.²³ A mixed model ANOVA was run to determine if there was a difference between ESQ-14 scores for before HA, after HA, and +25d. A Pearson's product-moment correlation was run to assess the relationship between delta ESQ-14 scores and delta HR, T_{rec} , T_{sk} , OMNI, and thermal sensation. Delta ESQ-14 scores (IPE ESQ-14 – PRE ESQ-14) were calculated to account for variance among subjects (over responders and under-responders). Delta HR, T_{rec} , T_{sk} , OMNI, and thermal sensation was calculated as IPE - PRE measures for each trial. A linear regression was run to understand the effect of delta perceptual and physiological measures on the delta ESQ-14 scores during each trial. (Before HA, after HA, and +25d). Alpha level was set at 0.05 for all tests. Data are mean \pm standard deviation, and 95% confidence intervals (CI) included where applicable (M \pm SD [95% CI]), unless otherwise stated.

CHAPTER IV: RESULTS

Participant characteristics were as follows: age, 23 ± 3 y; height, 179 ± 6 cm; weight, 73.47 ± 7.71 kg; percent body fat, $7.6 \pm 4.8\%$; $\text{VO}_{2\text{max}}$, 55.1 ± 5.7 ml·kg⁻¹·min⁻¹. Heat acclimation was confirmed with HR, T_{rec} , SR, and exercise time (table 1). Immediately post exercise HR and T_{rec} were significantly lower after HA (16 ± 1 bpm and $0.58 \pm 0.02^\circ\text{C}$, respectively). Additionally SR increased after HA compared to before HA by 0.29 ± 0.08 L·hr⁻¹. Participants were able to exercise for a longer duration (~ 8.06 minutes) following the HA protocol compared to before HA.

Table 1. Physiological response (immediately post exercise) before and after heat acclimation. (Mean \pm SD)

	Before HA	After HA
Heart Rate (bpm)	158 ± 19	$143 \pm 18^*$
Rectal Temperature ($^\circ\text{C}$)	$39.25 \pm .72$	$38.71 \pm .64^*$
Sweat Rate (L·hr⁻¹)	$-1.50 \pm .45$	$-1.93 \pm .48^*$
Exercise Time (min)	109.20 ± 14.97	$118.00 \pm 6.28^*$

Note: Data was pooled for before and after HA for analysis (n=15).

Abbrev: HA, heat acclimation.

*Indicates significance from before to after HA ($p < 0.001$).

Average IPE ESQ-14 score before HA (22 ± 8 [16, 28]) and after HA (11 ± 7 [6, 17]) was significantly greater than average PRE ESQ-14 score before HA (5 ± 3 [2, 7]) and after HA (5 ± 3 [2, 7]) (all, $p < 0.001$) (figure 2). Importantly, HA elicited a statistically significant mean decrease in delta ESQ-14 scores after HA compared to before HA (10 ± 6 [6, 13]), $t(14) = 5.895$, $p < 0.001$ (figure 3). However, no significant differences were found between groups (IHE and NHE) and day (After HA and +25d) when comparing delta ESQ-14 scores, $p = 0.467$ (figure 4). Table 2 and 3 represents raw data for PRE, IPE, and delta ESQ-14 scores.

The NHE group showed a greater loss of adaptations (HR: 163%; T_{rec} : 87%) compared to IHE (HR: 53%; T_{rec} : 2.7%) on +25d. The IHE group sustained physiological adaptations better than the NHE group by performing exercise in the heat every 5th day.

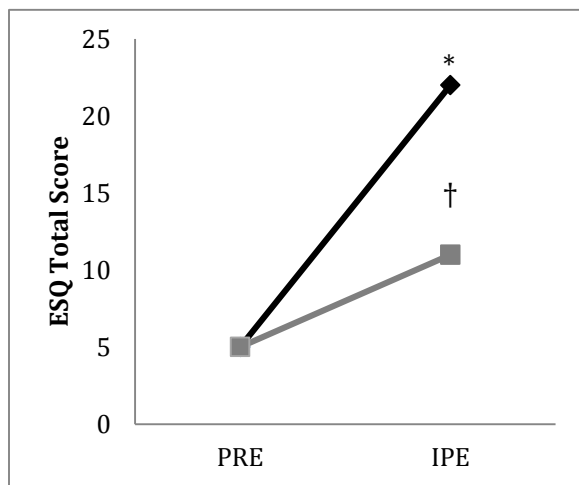


Figure 2. Environmental symptoms questionnaire total scores pre exercise and immediately post exercise before (black) and after (gray) heat acclimation; n=15. Abbrev: ESQ, environmental symptoms questionnaire; PRE, pre exercise; IPE, immediately post exercise; HA, heat acclimation. *Indicates a significant difference between PRE and IPE ($p<0.001$). †Indicates a significant difference between IPE before and after HA ($p<0.001$).

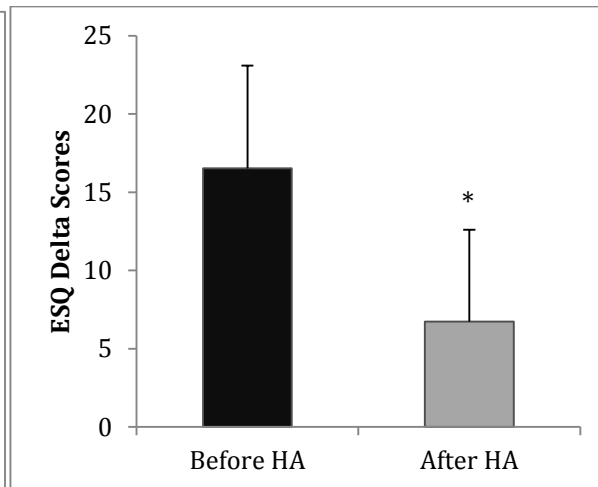


Figure 3. Environmental symptoms questionnaire delta scores (IPE-PRE) before (black) and after (gray) heat acclimation; n=15. Abbrev: ESQ, environmental symptoms questionnaire; HA, heat acclimation; IPE, immediately post exercise; PRE, pre exercise. *Indicates a significant mean difference between days ($p<0.001$).

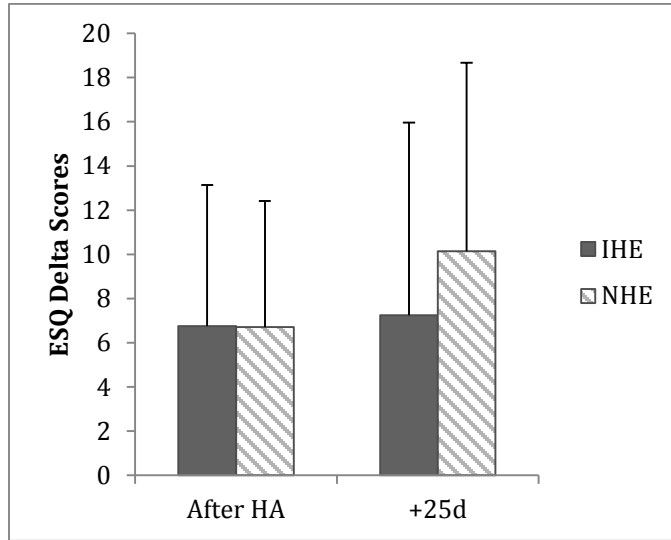


Figure 4. Environmental symptoms questionnaire delta scores (IPE-PRE) after HA and +25d for intermittent heat exposure (n=8; solid) and no heat exposure (n=7; cross hatch). Abbrev: ESQ, environmental symptoms questionnaire; HA, heat acclimation; IPE, immediately post exercise; PRE, pre exercise, IHE, intermittent heat exposure; NHE, no heat exposure. No significant differences between day and group (p=0.476).

Table 2. Average environmental symptom questionnaire total scores before and after HA. (Mean ± SD)

Day	PRE Exercise Total	IPE Exercise Total	Delta Score (IPE-PRE)
Before HA	5 ± 3	22 ± 8*	17 ± 7
After HA	5 ± 3	11 ± 7*†	7 ± 6†

Note: Data was pooled for before and after HA for analysis (n=15).

Abbrev: ESQ, environmental symptoms questionnaire; HA, heat acclimation; PRE, pre exercise; IPE, immediately post exercise.

*Indicates significance from PRE to IPE (p<0.001).

†Indicates significance from before to after HA (p<0.001).

Table 3. Average environmental symptom questionnaire total scores following heat acclimation. (Mean \pm SD)

Day	Group	PRE Exercise Total	IPE Exercise Total	Delta Score (IPE-PRE)
After HA	NHE	7 \pm 4	14 \pm 7*	7 \pm 6
	IHE	3 \pm 2	10 \pm 6*	7 \pm 6
+25d	NHE	6 \pm 3	16 \pm 8*	10 \pm 9
	IHE	4 \pm 3	11 \pm 8*	7 \pm 9

NHE: No heat exposure (n=7); IHE: Intermittent heat exposure (n=8).

Abbrev: ESQ, environmental symptoms questionnaire; HA, heat acclimation, PRE, pre exercise; IPE, immediately post exercise.

*Indicates significance from PRE to IPE (p<0.001).

The most frequently reported symptoms throughout the study for PRE and IPE measures are reported in table 4; maximal number of responses is from all participants (n=15). The most frequently report symptom for PRE and IPE exercise throughout all trials was “I feel hot.”

Table 4. Top 5 most reported symptoms pre exercise and immediately post exercise (Max number of responses= 15).

PRE		IPE	
“I feel hot”	14	“I feel hot”	14
“I feel tired”	12	“I feel weak”	14
“I will play at my best”	11	“I will play at my best”	14
“I feel thirsty”	10	“I feel thirsty”	13
“I feel weak”	5	“I feel tired”	13

Abbrev: PRE, pre exercise; IPE, immediately post exercise.

The correlation of delta ESQ-14 scores to delta physiological and perceptual measures during exercise are represented in table 5. Before HA physiological and perceptual measures were not significantly correlated to the delta ESQ-14 scores, however, OMNI scale and thermal sensation scale did approach significance (p=0.065, p=0.086, respectively). There was a statistically significant relationship between T_{rec} , OMNI scale, and thermal sensation scale to delta ESQ-14 scores after HA (p=0.026, p=0.005, p=0.001, respectively). Additionally, we found

a statistically significant correlation between T_{rec} , HR, and OMNI and delta ESQ-14 scores +25d ($p=0.018$, $p=0.021$, $p=0.001$, respectively).

Table 5. Delta (IPE-PRE) exercise Pearson correlation (r) and r^2 between ESQ-14 and physiological and perceptual measures.

Variables	ESQ-14		
	Before HA r (r^2)	After HA r (r^2)	+25d r (r^2)
Heart Rate	0.180 (0.032)	0.471 (0.221)	0.589 (0.347)*
Rectal Temperature	-0.085 (0.007)	0.573 (0.328)*	0.600 (0.36)*
Skin Temperature	0.217 (0.047)	0.291 (0.085)	-0.401 (0.161)
OMNI Scale	0.487 (0.237)	0.707 (0.5)*	0.767 (0.588)*
Thermal Sensation	0.457 (0.209)	0.798 (0.637)*	0.258 (0.067)

Abbrev: ESQ, environmental symptoms questionnaire; HA, heat acclimation; PRE, pre exercise; IPE, immediately post exercise.

*Indicates correlation at the $P \leq 0.05$ level (2 tailed).

Significant variables were retained in the final linear regression model for after HA (T_{rec} , OMNI, and thermal sensation) and +25d (T_{rec} , HR, and OMNI). A linear regression established that change in thermal sensation accounted for 63.6% of the explained variability in delta ESQ-14 scores after HA ($r^2=0.636$, $p<0.001$ [4,11]). Additionally, a linear regression established that change in OMNI accounted for 58.9% of the explained variability in delta ESQ-14 scores after the maintenance and decay of HA (+25d) ($r^2=0.589$, $p=0.001$ [95% CI, 1-4]).

CHAPTER V: DISCUSSION

Heat acclimation is an extremely efficient way to reduce physiological and psychological strain from exercising in the heat. Heat acclimatization has proven to reduce thermal strain through physiological adaptations.^{1,18} Not only did participants adapt physiologically but they also experienced a decrease in symptom severity once HA, as evident from our findings. Previous research has shown that the delta ESQ-14 scores began to decrease within four days of a 10-day HA protocol and retained this decrease throughout the remainder of HA.⁹ Additionally, Johnson et al¹² found that the total score of symptom severity was significantly higher during the first 2 days of HA compared to the remaining 8 days of their HA protocol. Our findings were similar in that a 10-day HA protocol significantly decreases overall symptom severity, which is evident through a decrease in delta ESQ after HA. However, our study did not measure ESQ-14 daily throughout the HA protocol. After a bout of exercise in the heat participants experienced significantly greater symptoms compared to the start of exercise. The increase in symptom severity after exercise was evident regardless of HA status, however HA did decrease the extent of symptom severity.

The present study was the first study to our knowledge that explored the ESQ-14 during the decay and maintenance of HA. Once thermal stimulus is removed the decay of adaptations begins. A heat exposure every 5th day assisted in the maintenance of HA adaptations, as well as, the maintenance of ESQ-14 scores. Individuals who did not receive a thermal stimulus for 25 days after HA did not experience greater symptom severity compared to individuals who received a heat exposure every 5th day. Over the 25-day period neither groups delta ESQ-14 scores returned to their before HA values. Therefore, under extreme environmental conditions individuals who are HA perceived less symptom severity, regardless of the percentage loss of

physiological adaptations. Nevertheless, further research is needed to determine the length of time it takes for psychological response to return to a pre HA state.

Both groups behaved similarly throughout the study, which may also explain why we found no significant difference between IHE and NHE. We experienced some participants who were non-responders in both groups; who did not perceive high heat stress, while physiological strain was present as evidence by increased T_{rec} and HR. An example of this is through two specific cases (X and Y). Prior to HA participant X and Y were unable to complete the 120 minute HST (114 and 117 minutes, respectively). Both participants T_{rec} reached $\geq 39.99^{\circ}\text{C}$ and HR was 171 (X) and 188 (Y). However, the ESQ-14 delta score for participant X and Y were 10 and 24, respectively. This example demonstrates the importance of taking objective measures and not relying on subjective responses to determine thermal strain or to diagnose EHI. When obtaining subjective reactions we have to remember that there may be personal bias from the participant, and that individual perception varies among individuals.

Another subjective tool commonly used in research is the ratings of perceived exertion (RPE) scale; within the study, we did not use RPE, instead we used the OMNI scale, which is a similar measurement of perceived exertion.²¹ Physiological variables account for 60% of the variability in RPE; the other 40% of variance can be influenced by psychological factors.²⁴ Previous research found that individuals who are depressed, neurotic, or anxious have difficulty processing perceptual information.²⁴ Researchers and clinicians should remember that measurement of the ESQ-14 or other subjective scales is limited by the participant motivation and attentiveness, and potentially psychological states and traits.^{6,24,25} Therefore the influence of cognitive mediation must be taken into account when assessing variance of a subjective measure.

The participants were college age males and the study took place between the fall and spring semester. It is speculated that school (i.e., finals week) and outside factors (i.e., work and sleep) could affect the reported symptoms and the total score of the ESQ-14. Armstrong et al²⁶, found that the most frequent predisposing factors or warning signs of exertional heat illness were sleep loss and generalized fatigue. Our findings showed that “I feel tired” was within the top five most frequently reported symptoms before and after exercise, showing that individuals arrived feeling tired. In addition we found that of the most reported symptoms were feeling hot, tired and weak. Similarly, Johnson et al¹² found that the predominant symptoms during HA were warmth, sweatiness, and weakness. We speculated that these symptoms were driven by physiological and cardiovascular strain.

To determine what causes the change in perceptual ratings we explored the variance that can be explained by physiological adaptations. Stearns et al⁹ found a significant correlation of the modified ESQ-14 and HR ($r^2=0.124$), however the modified ESQ-14 was not significantly correlated with T_{rec} ($r^2=0.062$). In comparison our findings showed a correlation with T_{rec} after HA ($r^2=0.328$) and HR +25d ($r^2=0.347$). Although the findings showed a significant correlation, they are not clinically relevant. Stearns et al⁹ also examined the correlation of the ESQ-14 to RPE and found no significant correlation ($r^2=0.091$). In contrast, our study found that the OMNI and thermal sensation scale accounted for 63.6% of the variability in the ESQ-14 after HA. This study was the first to our knowledge to explore the relationship between thermal sensation and OMNI scale to the ESQ-14.

Although the thermal sensation scale was significantly correlated to the ESQ-14, T_{sk} did not reveal a relationship with the ESQ-14. It is speculated that the thermal sensation scale increased responses of “I feel hot” because it was the most reported symptom throughout all

trials. It has been suggested that thermal sensation is dependent on skin temperature due to an increase in peripheral circulation and efferent activity.²² Further research is needed to determine the relationship between T_{sk} and the ESQ-14, as our findings did not fully support this theory.

The ESQ-14 can provide a clinician with a subjective report of heat illness when, in certain circumstances, physiological measures are not available. However it is important to obtain objective measures (T_{rec}) to evaluate the severity of heat illness (i.e., heat exhaustion vs. exertional heat stroke). Physiological measures did not explain the variance in the ESQ-14, therefore it is imperative that clinicians obtain objective measures when diagnosing EHI. In a clinical setting physiological variables are measured during an emergency situation but not always measured during a HA protocol. Further research is needed to explore the use of the ESQ-14 during a HA protocol in a control and clinical setting, where temperatures may fluctuate day to day.

Limitations

The methodology of this study contained limitations. First, this study took place in an environmental chamber where temperature was carefully controlled; however in a clinical setting environmental conditions vary and can affect the thermal strain placed on an individual. Secondly, there are various factors that affect HA (i.e., gender, age, and fitness level); participants were physically fit college age males. Thirdly, our study was limited by the small sample size.

Future directions

The outcome of this study indicates that further research is needed to determine the use of the ESQ-14 within a clinical setting. Future direction should include the decay of subjective reports of heat illness to determine how long these adaptations last and if participants will perceive

heat strain similar to their pre HA status. It is clear from our study that reports vary among individuals who are undergoing similar thermal strain. Therefore, further research is needed to determine the affect of various personality traits on subjective reports during heat exposure in a laboratory and clinical setting.

Conclusion

Our study was the first, to our knowledge, to assess the ESQ-14 during the decay and maintenance of HA. Additionally, this study was the first to explore the relationship between the ESQ-14 and T_{sk} , OMNI, and thermal sensation scale. Heat acclimated individuals perceived and incurred less physiological heat stress compared to their pre-acclimated state. The ESQ-14 assessed overall HA induction, through the reduction of symptom severity, and verified some (T_{rec}), but not all (HR) HA induction responses. Therefore, the ESQ-14 may not be useful in predicting specific HA adaptations, but is useful in assessing overall HA induction due to the reduction of symptom severity. To ensure full physiological adaptations the ESQ-14 should be used in combination with physiological variables, such as HR and T_{rec} , to help confirm a decrease in thermal strain; this will ensure that athletes, military personnel, and laborers are safe while performing in a hot environment. Athletic trainers and clinicians can use the ESQ-14 to assess subjective responses to HA, however it does not replace objective measures such as HR and T_{rec} .

Further research is needed to determine the use of the ESQ-14 during the decay and maintenance of physiological adaptations. The ESQ-14 should not be used to determine if an individual is safe to return to physical activity after a period no heat exposure. Therefore, an individual must rely on physiological measures when choosing a HA maintenance plan to ensure they are returning to the heat safely.

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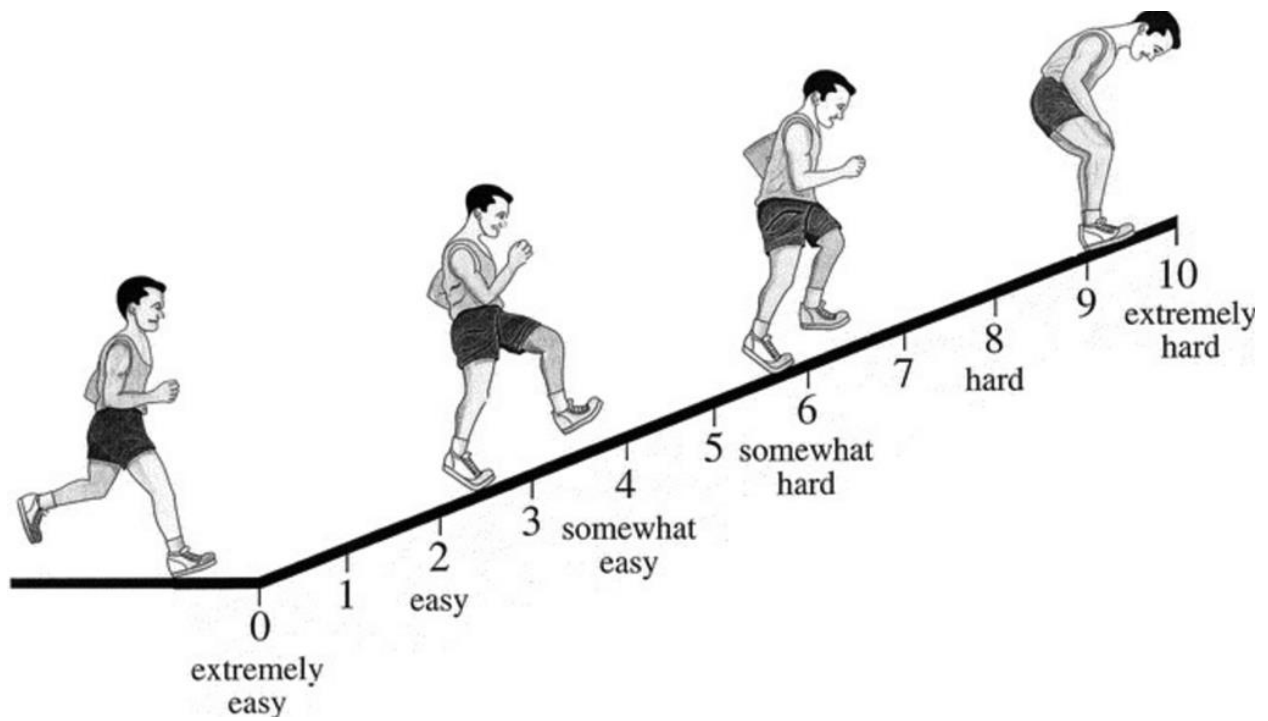
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APPENDICES

Appendix A

OMNI Scale



Appendix B

Thermal Scale

- | | |
|----------|------------------------|
| 0 | Unbearably Cold |
| 1 | Very Cold |
| 2 | Cold |
| 3 | Cool |
| 4 | Comfortable |
| 5 | Warm |
| 6 | Hot |
| 7 | Very Hot |
| 8 | Unbearably Hot |

Appendix C

Environmental Symptoms Questionnaire

Subject #: _____

Date: _____

How Do You Feel Questionnaire

1. Place an X in the box to explain HOW YOU HAVE BEEN FEELING TODAY.
2. PLEASE ANSWER EVERY ITEM.
3. If you did not have the symptom, say NOT AT ALL.

Symptoms	Not At All	A Little	Somewhat	Moderate	A Lot	Extreme
I feel lightheaded						
I have a headache						
I feel dizzy						
I feel thirsty						
I feel weak						
I feel grumpy						
It is hard to breathe						
I will play at my best						
I have a muscle cramp						
I feel tired						
I feel sick to my stomach (nauseous)						
I feel hot						
I have trouble concentrating						
I have "goose bumps" or chills						

SOURCE: Modified from Kobrick and Sampson (1979) and Sampson and Kobrick (1980).